Fate of Avermectin B_{1a} on Citrus Fruits. 2. Distribution and Magnitude of the Avermectin B_{1a} and ^{14}C Residue on Fruits from a Picked Fruit Study

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Avermectin B_{1a}, labeled with ^3H or ^{14}C, was applied to untreated, picked oranges. A migration of the radiolabeled residue from the fruit surface into the flavedo rind occurred rapidly within the first week postapplication and slower thereafter. Most of the rind radiolabeled residue was extractable by acetone at all time periods postapplication. The amount of avermectin B_{1a} was greater in the rind extract than the fruit surface rinse at 1 and 2 weeks postapplication. The pulp radiolabeled residues were very low (approximately 2 ppb) as were the unextractable rind radiolabeled residues at the field application rate. The dissipation of avermectin B_{1a} was biphasic with a rapid loss (78–96%) within 1 week ($t_{1/2} < 7$ days) and a slower loss from 1 to 4 weeks ($t_{1/2} = 20$ days) postapplication. No differences in the fate of avermectin B_{1a} were observed between the application rates. No differences were observed between [^3H]- and [^{14}C]avermectin B_{1a}, demonstrating the validity of using ^3H-labeled avermectin B_{1a} for crop metabolism studies. The fate of avermectin B_{1a} on picked orange fruit was similar to the fate of avermectin B_{1a} on field orange fruits.

The avermectins, a newly discovered class of pesticidal agents, are macrocyclic lactones produced by the actinomycete *Streptomyces avermitilis* (Burg et al., 1979). The avermectin structures have been elucidated (Albers-Schonberg et al., 1981), and some of the biological activities have been reported (Campbell et al., 1983). Abamectin (avermectin B_1 or MK-0936) is the commercial product that is undergoing EPA registration as an acaricide for citrus crops. Abamectin consists of ≥80% avermectin B_{1a} and ≤20% avermectin B_{1b} (Figure 1).

A previous field citrus metabolism study on oranges and lemons was conducted using [^3H]avermectin B_{1a} at approximately the field application rate (Iwata et al., 1985). From this study, preliminary information on the fate of avermectin B_{1a} on citrus fruits was obtained. In order to obtain more definitive information, a second field study on oranges, grapefruit, and lemons was conducted using [^{14}C]avermectin B_{1a} (Maynard et al., 1989). Since field studies can potentially produce results that can be affected by environmental parameters, the fate of avermectin B_{1a} on picked oranges was investigated.

The purpose of the picked orange fruit studies reported here was to investigate the fate of avermectin B_{1a} on picked fruits and compare the results to field fruit studies. If similar, the picked fruit procedure would provide a valid system to investigate the fate of avermectin B_{1a} or possibly other pesticides on citrus fruits. This report summarizes the results from the picked orange fruit studies and compares them to the results from field orange fruit studies. Furthermore, this report summarizes the fate of avermectin B_{1a} as compared between ^3H- and ^{14}C-labeled avermectin B_{1a} and 1×, 10×, and 25× application rates for both picked and field fruit studies.

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MATERIALS AND METHODS

The citrus fruits used for these studies were mature Valencia oranges from the University of California, CA, or nearly mature Valencia oranges from the University of Florida, Lake Alfred, FL. All oranges had not been previously sprayed with any chemical. Oranges were picked, shipped immediately to our laboratories in Rahway, NJ, and refrigerated upon arrival. The fruits were grouped indiscriminately, rinsed with water, and weighed. Application was made by completely wetting the oranges with the treatment solution with a glass pipet and collecting the runoff. The volume and concentration of the treatment solution were measured before and after treatment. Each orange received approximately 0.5–0.6 mL of treatment solution. After most of the treated orange was dry (10–20 min), each was placed on a separate glass Petri dish. All treated oranges were placed in a ventilated treatment box and placed outside. Orange samples had a plastic cover to protect the fruit from rain. This cover did not block incident light above 220 nm as measured by a UV/vis spectrophotometer.

Control oranges, treated with formulation containing no avermectin B_{1a}, were run at the same time and sampled accordingly. Treatment solutions of avermectin B_{1a} were made at approximately the field application rate (0.025 lb of ai/500 gal per acre) and at rates 10 and 25 times greater. A group of oranges (three or six) was sampled at various times (0, 1, 2, 4 weeks) after application and processed as a composite sample according to the procedure outlined in Figure 2. The radioactivity in the surface rinses and the rind extracts were determined by direct liquid scintillation counting (LSC) of aliquots. The remaining fractions were homogenized, and subsamples were combusted and counted for radioactivity by LSC. Specific differences between the studies will be described below.

Treatment Solutions. (1) [5-^3H]Avermectin B_{1a} (48 μg; 8 ppm ^3H) and 15 μL of soluble liquid (SL) formulation (witconol/propylene glycol, 12/88, w/w) were diluted to 6 mL with deionized distilled water. The specific activity of the [^3H]avermectin B_{1a} was 118 μCi/mg with a 98+% radiochemical purity. (2) [3,7,11,13,23-^{14}C]Avermectin B_{1a} (113 μg; 8 ppm ^{14}C) and 35 μL of formulation were diluted to 14 mL with deionized distilled water. The specific

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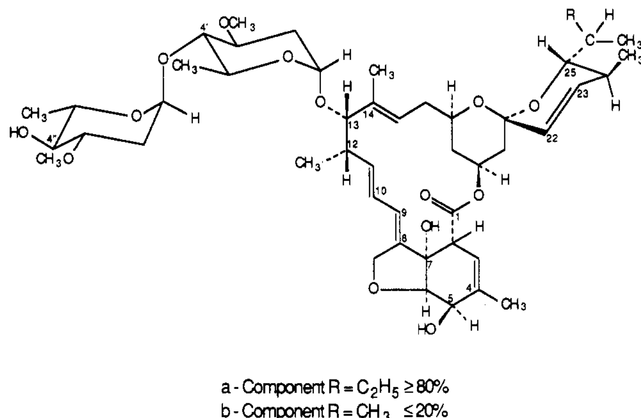

Figure 1. Structures of the two major components of abamectin.

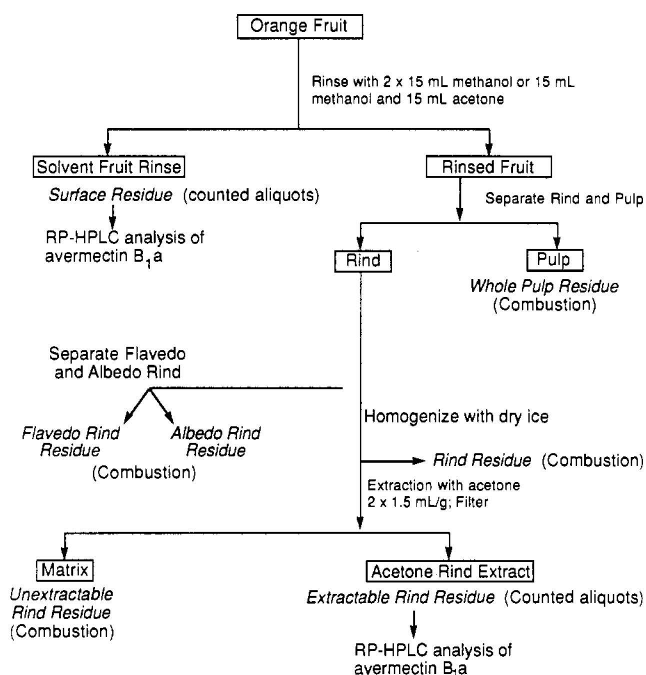

Figure 2. Orange fruit processing procedure.

activity of the [^{14}C]avermectin B_{1a} was 16.4 μCi/mg with a 99+% radiochemical purity. (3) [5-^{3}H]Avermectin B_{1a} (1.44 mg; 80 ppm ^{3}H) and 400 μL of formulation were diluted to 18 mL with distilled water. The specific activity of the [^{3}H]avermectin B_{1a} was 103 μCi/mg with a 98+% radiochemical purity. (4) [3,7,11,13,23-^{14}C]Avermectin B_{1a} (4.77 mg; 200 ppm ^{14}C) and 1.38 mL of formulation were diluted to 24 mL with deionized, distilled water. The specific activity and radiochemical purity were the same as treatment solution 2.

Individual Studies. (1) Six oranges (two groups of three) were treated with the 8 ppm ^{3}H solution, and one control orange was treated with blank formulation on 3 Aug 1982 in New Jersey. A group was processed at 0 (30–45 min) and 2 weeks after treatment according to the procedure outlined in Figure 2. (2) Twelve oranges (four groups of three) were treated with the 8 ppm ^{14}C solution, and two controls were treated with blank formulation and started on 3 Aug 1982 in New Jersey. A group was processed at 0, 1, 2, and 4 weeks after treatment. (3) Twenty-four oranges (four groups of six) were treated with the 80 ppm ^{3}H solution, and six controls were treated with blank formulation and started on 14 Sept 1981 in New Jersey. After drying, the treated oranges were placed on Petri dishes outside with a plastic wrap covering above the fruit. The plastic wrap was shown to not block light above 220 nm as measured by a UV/vis spectrophotometer. A group was processed at 0, 1, 2, and 4 weeks after treatment. In this study, the fruit were rinsed with 2 × 15 mL of methanol; this rinse represented the surface residue fraction. Also, the rind was visually separated into outer (flavedo) and inner (albedo) rind fractions. (4) Thirty-three oranges (three groups) were treated with the 200 ppm ^{14}C solution, and of seven controls, four were treated with blank formulation and three were untreated and started on 3 Aug 1982 in New Jersey. A group of three oranges was sampled at 0 and 1 week, and a group of 27 oranges was sampled at 2 weeks after treatment. In addition, the rinds were further extracted by homogenization with acetone (1.5 mL/g of rind). The spend rind solid (matrix) was separated from the acetone extract by filtration. Furthermore, for this 200 ppm study, most of the treated fruit, half of the treated controls, and none of the untreated controls exhibited a browning of the rind at 2 weeks. This effect was attributed to a formulation dependent change that did not appear to affect the results.

Avermectin B_{1a} Determination. The amount of avermectin B_{1a} in the radiolabeled residue that was extractable from the oranges was determined. The methanol fruit rinses for all studies and the acetone rind extracts for the 25×-treated orange study were concentrated under a stream of N_2 and analyzed by RP-HPLC. The amount of radioactivity eluting as a definitive peak at the retention time of an avermectin B_{1a} standard was determined and expressed as a percent of the total radioactivity eluting from the LC column. This value represents the fraction of avermectin B_{1a} in the surface rinse or the rind extract. The fraction of avermectin B_{1a} in the extract multiplied times the concentration of the radiolabeled residue in the extract, expressed as nanogram equivalents per gram of whole fruit, gave the nanograms of avermectin B_{1a} per gram of whole fruit (ppb). Duplicate injections of a sample gave reproducible percent values for avermectin B_{1a}. The radioactivity eluting through the LC column was 85–97% of that present in the various extracts analyzed. For confirmation, the radioactivity eluting at the retention time of avermectin B_{1a} for one sample was collected and injected on a different RP-HPLC system. The radioactivity attributed to avermectin B_{1a} from the first LC system eluted at the retention time of an avermectin B_{1a} standard on the second system. The RP-HPLC conditions used for the determination of the level of avermectin B_{1a} in the fruit extracts are described below. A 100% CH_3OH column wash was collected after the isocratic run for accurate recovery determination.

Instrumentation. *High-Performance Liquid Chromatography (HPLC).* An HPLC system was used with a 4.6 mm × 250 mm DuPont Zorbax ODS column and a precolumn containing Whatman Pell ODS packing. The mobile phase was 85/15 (v/v) CH_3OH/H_2O, and the flow was 1.0 mL/min. The column effluent was monitored at 245 or 254 nm, and 1-min fractions were collected and counted for radioactivity.

Liquid Scintillation Counting (LSC). A Packard instrument (Packard, United Technologies, Inc.) was used. Samples were counted in Insta-gel (Packard, United Technologies, Inc.), and dpm values were obtained from appropriate quench curves.

Sample Combustion. A Packard instrument (Packard, United Technologies, Inc.) was used for sample combustion. Tritium was collected as ^3H_2O in Monophase 40 (Packard, United Technologies, Inc.) and carbon-14 as $^{14}CO_2$ in Carbosorb and Permafluor V (Packard, United Technologies, Inc.); dpm values were determined by LSC

Table I. Total Radioactivity in the Fruit Fractions as a Percent of the Total Initial Radioactivity[a]

treatment dose	time, weeks	surface rinse	whole rind	whole pulp
8 ppm/^{3}H	0	95 (95)	5 (5)	<DL
	2	16 (24)	46 (70)	4 (6)
8 ppm/^{14}C (1×)	0	96 (96)	4 (4)	<DL
	1	60 (44)	39 (40)	<DL
	2	38 (46)	44 (54)	<DL
	4	42 (42)	55 (55)	2 (2)
80 ppm/^{3}H (10×)	0	87 (87)	13 (13)	<0.1 (<0.1)
	1	51 (56)	39 (43)	1 (1)
	2	29 (41)	45 (63)	2 (2)
	4	28 (38)	45 (60)	2 (2)
200 ppm/^{14}C (25×)	0	88 (88)	9 (9)	3 (3)
	1	22 (29)	49 (66)	3 (5)
	2	17 (20)	65 (73)	7 (7)

[a] Values in parentheses are expressed as a percentage of the total radiolabeled residue recovered for the sample. DL = detection limit. Reported values are the mean of triplicate measurement of composite samples (three or six oranges).

using appropriate quench curves.

Materials. The [^{3}H]avermectin B_{1a} was obtained in house from the Labeled Compound Synthesis Group, Rahway, NJ, and the [^{14}C]avermectin B_{1a} was synthesized as previously described (Ku et al., 1985). All formulations were standards obtained in house from the Agricultural Formulations Group, Three Bridges, NJ. All other chemicals used were reagent grade or better, and solvents were HPLC grade or better. All materials were purchased from commercial suppliers.

RESULTS

Residue Levels and Distribution. The total radioactivity in the various fruit fractions was determined by LSC and expressed as a percentage of the total initial radioactivity determined at zero time (Table I) or as nanograms of avermectin B_{1a} equivalents per gram of initial whole fruit (ppb) (Table II).

For all application levels 87–96% of the total radioactivity was recovered in the methanol surface rinse at zero time (Table I). The radioactivity recovered in the rind for the zero time fruit was 4–13% of the total radioactivity with the larger percentages observed at the higher application rates. At 1 week postapplication, the values of radioactivity recovered in the fruit-surface rinse for the 1×-, 10×-, and 25×-treated oranges were 60, 51, and 22% of the total initial radioactivity, respectively (Table I). The corresponding values of radioactivity in the rinds were 39, 39, and 49% of the total initial radioactivity, respectively. At 1 week for the oranges treated at 1× and 10×, the percentage of the total initial radioactivity was greater in the fruit-surface rinse than the rind. For the 25× oranges at 1 week, the opposite was observed. At 2 and 4 weeks postapplication for all application rates, the percentage of the total initial radioactivity was greater in the rind than the corresponding fruit-surface rinse (Table I). The rate of migration of the radioactivity from the fruit-surface into the rind was greater within 1 week than after 1 week postapplication for all studies. For example, at all application rates the increase in radioactivity in the rind was 26–40% and 6–16% of the total initial radioactivity for 0 to 1 weeks and 2–4 weeks postapplication, respectively. Also, the decrease in the fruit-surface rinse was 36–66% and 18–23% of the total initial radioactivity for 0 to 1 weeks and 2–4 weeks, respectively (Table I).

The total radiolabeled residues in the various fruit fractions from these studies were expressed as nanograms of avermectin B_{1a} equivalents per gram of initial whole fruit (ppb) and are presented in Table II. The initial residues (total zero time radiolabeled residue) were 28–37, 116, and 527 ppb for the 1×, 10×, and 25× application rates, respectively. The total radiolabeled residue for the 1×-treated ^{3}H and ^{14}C oranges was the same at 2 weeks postapplication (24 vs 23 ppb). The recoveries of the total radiolabeled residue for all these studies were observed to remain the same or decrease slightly with time postapplication.

The radiolabeled residues in the rind samples for these studies were determined by combustion and expressed for comparison as nanograms of avermectin B_{1a} equivalents per gram of initial whole fruit weight. However, the actual rind radiolabeled residue levels are approximately 4 times higher on the basis of the weight of the rind fraction. After the whole fruit was rinsed with methanol to remove the surface residues, the radiolabeled residues in the rind from the 10×-treated oranges were investigated by sampling the outer (flavedo) and inner (albedo) rind fractions. Most (90–93%) of the radiolabeled residue in the rind was located in the flavedo rind fraction (Table II). Also, the flavedo to albedo residue ratio remained constant from 1 to 4 weeks.

The amounts of radiolabeled residue in the whole pulp fractions from oranges treated at the approximate field application rate were 0.6 and 1.5 ppb in two of six samples that contained detectable residues (Table II). Actual levels in the pulp were 0.7–1.8 ppb. For all application rates, the levels of radioactivity in the whole pulp fractions as a percentage of the total initial radioactivity were similar

Table II. Levels of the Radiolabeled Residue in the Fruit Fractions Expressed as Nanogram of Avermectin B_{1a} Equivalents per Gram of Total Initial Whole Fruit[a]

treatment	time, weeks	MeOH rinse	acetone rinse	flavedo rind[b]	albedo rind[b]	whole rind[b]	whole pulp[c]	total
8 ppm/^{3}H	0	33.0	2.3			1.9	<DL	37.2
	2	5.5	0.4			17.0	1.5	24.4
8 ppm/^{14}C (1×)	0	25.4	1.4			1.1	<DL	27.9
	1	13.0	3.7			11.0	<DL	27.7
	2	7.5	3.0			12.3	<DL	22.8
	4	9.3	2.5			15.4	0.6	27.8
80 ppm/^{3}H (10×)	0	101.3		13.5	1.5		0.1	116.4
	1	59.0		42.5	3.4		1.3	106.2
	2	33.9		48.9	3.6		1.9	88.3
	4	33.1		48.4	3.8		1.9	87.2
200 ppm/^{14}C (25×)	0	432.2	29.7			47.3	17.7	526.6
	1	77.8	37.0			258.4	18.3	391.5
	2	67.5	23.9			340.1	34.4	466.8

[a] If no value reported, sample not applicable. Reported values are the mean of triplicate measurement of composite samples (three or six oranges). [b] Actual rind radiolabeled residue levels are approximately 4 times higher based on rind weight. [c] Actual pulp radiolabeled residue values are approximately 1.2 times higher based on pulp weight.

Table III. Extraction of the Rind Radiolabeled Residues with Acetone[a]

sample	whole rind, ng/g	acetone extract, ng/g	matrix, ng/g
0 week	47.3	27.4 (79)	7.2 (21)
1 week	258.4	125.3 (52)	117.8 (48)
2 week	340.1	172.4 (57)	132.5 (43)

[a] Values in parentheses are expressed as a percentage of the total radiolabeled residue recovered for the extraction procedure. Levels are reported as nanograms of avermectin in B_{1a} equivalents per gram of total initial fruit weight. Actual rind radiolabeled residue values are approximately 4 times higher based on rind weight. Actual matrix residue values are approximately 16 times higher based on the matrix weight. Reported values are the mean of triplicate measurement of composite samples of three oranges.

(Table I); however, the levels of the whole pulp radiolabeled residue were dependent on application rate and increased with time postapplication (Table II).

Extraction of the Rind Residue. After the whole fruit was rinsed with solvent to remove the surface residues, the rind from the 25×-treated oranges was removed and extracted with acetone. The percents of extractable and unextractable radiolabeled residue were determined (Table III). For 0–2 weeks postapplication, most (52–79%) of the rind radioactivity was extracted by acetone, resulting in 21–48% of the rind radioactivity remaining unextractable. Expressed as a percentage of the total initial radioactivity, the unextractable radioactivity in the rind by this procedure was 2, 24, and 28 for 0-, 1-, and 2-week samples, respectively. The greatest increase (22%) in the unextractable rind radioactivity was within the first week postapplication. The percent unextractable rind radioactivity increased only 4% from 1 to 2 weeks postapplication.

Determination of Avermectin B_{1a} Levels. The avermectin B_{1a} levels in the extractable residues from these studies were determined (Table IV). At zero time (30–45 min), the levels of avermectin B_{1a} were 54–75% of the total radioactive residue present. The levels of avermectin B_{1a} at 1 week postapplication were 8–14% of the total radioactive residue. In general, for all studies, the level of avermectin B_{1a} as a percent of the extract was similar for the corresponding methanol surface rinse samples. The levels of avermectin B_{1a} as a percent of the acetone rinse from the 1× and 25× studies were also similar for the corresponding samples for all studies (Table IV). For most samples, the level of avermectin B_{1a} as a percentage of the acetone rinse was slightly higher (1–6%) than the level of avermectin B_{1a} as a percent in the corresponding methanol rinse. The avermectin B_{1a} levels as a percent of the acetone rind extract for the 1- and 2-week, 25×-treated oranges were 39 and 36%, respectively (data not shown). For the 25×-treated oranges, the level of avermectin B_{1a} as a percent of the extract of the methanol rinse, acetone rinse, and acetone rind extract were 27, 31, and 39% for the 1-week oranges and 12, 17, and 36% for the 2-week oranges, respectively.

DISCUSSION

Picked Orange Studies. In all studies, the radiolabeled residues in the fruit-surface rinse declined rapidly within the first week postapplication and declined more slowly thereafter. The radiolabeled residues in the rind increased to near maximal levels within the first week postapplication. This indicated a fairly rapid migration of the radiolabeled residue from the fruit surface into the rind. Because this migration of the residue was observed in all studies reported here, it appears that the effect of dose or radiochemical label on the radiolabeled residue distribution was minimal. However, the migration of the residue into the rind appeared more extensive for the 25×-treated oranges.

The rind radiolabeled residue was mostly (90–93%) found in the flavedo rind fraction. The ratio of the flavedo to albedo rind residue remained constant for 1–4 weeks postapplication, indicating that the radiolabeled residue in the flavedo rind did not partition further into the fruit. Most (52–79%) of the radiolabeled residue was extracted with acetone from the rind of the 25×-treated oranges, which represented a worst case situation. The unextractable rind radiolabeled residues remaining after acetone extraction were 2, 24, and 28% of the total initial radiolabeled residue for 0-, 1-, and 2-week samples, respectively. As observed with the fruit surface to rind migration of the radiolabeled residue, the greatest change in the unextractable rind residues occurred within the first week postapplication. Therefore, the rind radiolabeled residues were mostly extractable and the unextractable rind residue did not appreciably increase with time after 1 week. Furthermore, the unextractable rind residues translated to the field application rate would be very low (less than 5 ppb whole fruit or 20 ppb actual concentration) by this acetone extraction procedure. Moreover, exhaustive extraction procedures on comparable unextractable rind samples (e.g., Bligh–Dyer and Soxhlet extraction and acid hydrolysis) reduced the unextractable rind residues at least 67% for field oranges (Maynard et al., 1989).

The radioactivity recovered in the pulp (edible portion) fraction, as a percent of the total initial radioactivity, was

Table IV. Levels of Avermectin B_{1a} in the Fruit Surface Rinse

applied dose	time, weeks	MeOH rinse,[a] %	MeOH rinse,[b] ng/g	acetone rinse,[a] %	acetone rinse,[b] ng/g	total
8 ppm/^{3}H	0	75	25	78	2	27
	2	21	1	25	<1	1
8 ppm/^{14}C (1×)	0	78	20	72	1	21
	1	26	3	32	1	4
	2	22	2	22	1	3
	4	17	2	20	1	3
80 ppm/^{3}H (10×)	0	62	63			63
	1	24	14			14
	2	23	8			8
	4	24	8			8
200 ppm/^{14}C (25×)	0	80	346	79	23	369
	1	27	21	31	12	33
	2	12	8	17	4	12

[a] Percent values are the total radioactivity eluting as the avermectin B_{1a} peak as a percent of the total radioactivity eluting through the column. Recovery of radioactivity from the column was 85–97%. Values are a single measurement of the composite sample. [b] Values are the nanograms of avermectin B_{1a} per initial whole fruit weight. [c] If no value reported, sample not applicable.

similar for all studies. The levels of radiolabeled residue, however, were dose dependent and approximately 1 ppb at the field application. The pulp radiolabeled residue levels when extrapolated from the higher rates were also approximately 1 ppb.

For all studies, the initial levels of avermectin B_{1a} (zero time) were 54–75% of the total radioactivity present. This demonstrates a rapid degradation of avermectin B_{1a} when applied to the oranges. Within 1 week (first time point), an extensive degradation (78–94%) of avermectin B_{1a} was observed for all studies. This indicates a degradation half-life of less than 1 week for the initial phase of dissipation based on these data. The actual initial half-life is probably 1 day or less as was shown on cotton leaves (Bull et al., 1984). After 1 week the loss of avermectin B_{1a} was much slower, indicating a biphasic dissipation. Since the dissipation data for avermectin B_{1a} were similar to that of other pesticides on citrus (Gunther, 1969) and due to the limited amount of data in Table IV, it was assumed that the dissipation of avermectin B_{1a} followed first-order kinetics after 1 week. The half-lives of avermectin B_{1a} excluding the first week were 20 and 30 days for the 1× and 10× application rates, respectively. This suggests a similar rate of degradation of avermectin B_{1a} at both application rates.

To further understand the fate of avermectin B_{1a} on oranges, the extractable rind radiolabeled residues from the 25×-treated oranges were analyzed by HPLC. The rind radiolabeled residue contained higher percentages of avermectin B_{1a} than the fruit-surface radiolabeled residue at 1 and 2 weeks postapplication as presented in the Results. Furthermore, from these percent values the amounts of avermectin B_{1a} (ppb) were higher in the rind extract than the fruit-surface rinse. Moreover, from 1 to 2 weeks, the amount of avermectin B_{1a} decreased considerably in the fruit-surface residue while the amount in the rind residue increased. Therefore, since the amount of avermectin B_{1a} actually increased in the rind residue while the amount in the surface rinse decreased with time after 1 week, partitioning of avermectin B_{1a} in the rind appeared to provide protection from degradation. The long residual acaricidal activity of avermectin B_{1a} observed in the field (McCoy et al., 1982) can be explained by the protection of avermectin B_{1a} from degradation in the fruit flavedo rind. These results also indicate that the degradation of avermectin B_{1a} was occurring on the fruit surface.

Picked vs Field Orange Fruit. Treatment of picked or field oranges with formulated avermectin B_{1a} at the approximate field rate resulted in similar total initial residue levels (21–29 ppb) when extrapolated to the actual field rate, 0.025 lb of ai/500 gal per acre or 6 ppm (Iwata et al., 1985; Maynard et al., 1989). The total initial residue levels from the 10× application rate were also similar between picked and field oranges (116 vs 203 ppb). The amounts of avermectin B_{1a} in the surface residue at certain time periods after 1 week were very similar (1–3 ppb whole fruit concentration) for both picked and field (Iwata et al., 1985; Maynard et al., 1989) oranges at the approximate field application rate. Therefore, the amount of avermectin B_{1a} present was very similar at all time points after application for both picked and field oranges.

The amount of avermectin B_{1a} in the surface residue decreased rapidly within 1 week followed by a slower decline to 4 and 12 weeks for both picked (Table IV) and field oranges (Maynard et al., 1989), respectively. A decline in the avermectin B_{1a} levels at zero time (10–45 min) was also observed for both picked and field fruit. Therefore, a similar biphasic dissipation of avermectin B_{1a} on oranges was observed for both picked and field fruit. In the initial phase, the degradation half-life of avermectin B_{1a} was less than 1 week for both picked and field fruit application. As discussed, this value is probably closer to 1 day. The half-life of avermectin B_{1a} for the second slower phase (7–28 days postapplication) was 20 days for picked oranges (Table IV) and 17 days for field oranges (15–60 days postapplication) (Iwata et al., 1985) at the approximate field rate. Furthermore, from California field trials with oranges (Jenkins, J., personal communication), the half-life of avermectin B_{1a} from 1 to 28 days postapplication was 14–18 days. This indicates that the rate of degradation of avermectin B_{1a} was the same on picked oranges as observed on field oranges. Therefore, from the biphasic dissipation profiles, levels of avermectin B_{1a}, and the half-lives of avermectin B_{1a}, the fate of avermectin B_{1a} was the same on both picked and field orange fruit.

Some differences were observed between the picked and field orange studies. In particular within 4 weeks postapplication, the total radiolabeled residue levels decreased 0–34% on picked oranges while they decreased approximately 50% on the field oranges (Iwata et al., 1985; Maynard et al., 1989). The losses in the field study were not due to removal by rain or volatilization of avermectin B_{1a}, which has a vapor pressure of less than 10^{-9} Torr. These losses in the total residue from the field oranges were attributed to the formation of volatile degradents of avermectin B_{1a} occurring on the fruit surface (Maynard et al., 1989).

The degradation of avermectin B_{1a} was shown to occur with a half-life of approximately 18 h in aqueous solution or on soil exposed to sunlight (Ku et al., 1983). Also, avermectin B_{1a} was shown to degrade more extensively on sunlight-exposed than sunlight-protected picked oranges within 1 week postapplication (Maynard et al., 1983). Furthermore, an extensive loss of ^{14}C recovery (55%) occurred from a film of [^{14}C]avermectin B_{1a} applied to glass Petri dishes and exposed to a sunlamp for 6 days (Maynard and Gruber, unpublished data). Therefore, the degradation of avermectin B_{1a} resulted in the formation of volatile components. This degradation on glass dishes was enhanced by sunlight ($t_{1/2}$ = 5 h) compared to protection from light ($t_{1/2}$ = 2 days) (MacConnell and Demchak, personal communication). Since the degradation of avermectin B_{1a} was shown to be similar on picked and field oranges, the volatile degradents of avermectin B_{1a} apparently did not dissipate with the conditions of the picked orange procedure. This could be attributed to less ventilation of the picked oranges than oranges in a field situation, resulting in higher total residue levels for the picked oranges. Because of these higher total residue levels, more residue was able to migrigate into the rind as indicated by higher rind residue levels for the picked vs field oranges. Therefore, the higher total residue levels and the greater partitioning of the residue into the rind were the only differences observed between the picked and field orange studies and were attributed to the lack of removal of the volatile degradents. This, however, did not affect the rate of degradation of avermectin B_{1a} as discussed above. Therefore, the use of the picked citrus fruit procedure provided accurate and comparable results to the field fruit studies for the fate of avermectin B_{1a}.

Application Rate. The levels of the total radiolabeled residue and avermectin B_{1a} were shown to be dependent on application rate. In general, as a percentage of the total initial radiolabeled residue, the distribution of the radiolabeled residue among the fruit fractions was similar at the various application rates. This indicated that the parti-

tioning of the residue with time did not vary much with the rate of application. The degradation of avermectin B_{1a} was also not dependent on application rate since the levels of avermectin B_{1a} as a percent of the fruit surface radiolabeled residue were similar among the studies. Therefore, the levels of the total radiolabeled residue and avermectin B_{1a} were dose dependent while the radiolabeled residue distribution and degradation of avermectin B_{1a} were dose independent.

[^{3}H]- vs [^{14}C]Avermectin B_{1a}. The validity of using [^{3}H]avermectin B_{1a} on field citrus fruits was preliminarily discussed (Iwata et al., 1985). On picked oranges at the field rate, the amount of avermectin B_{1a} was very similar at 0 and 2 weeks for ^{3}H and ^{14}C applications considering the variability in treatments between groups. The radiolabeled residue distribution and the total radiolabeled residue levels were also similar with time postapplication. Moreover, the avermectin B_{1a} levels as a percent of the fruit-surface residue was the same, which would not occur if the ^{3}H label on the parent compound was labile. These same comparisons, except the levels of avermectin B_{1a} and total radiolabeled residue, were observed in the picked oranges between the 1× ^{14}C and the 10× ^{3}H study.

From the field orange studies at the approximate field application rate, the results between [^{3}H]avermectin B_{1a} (Iwata et al., 1985) and [^{14}C]avermectin B_{1a} (Maynard et al., 1989) were comparable. The following were similar between these studies at the field application rate: the loss of the amount of total radiolabeled residue amounts with time, initial radiolabeled residue levels, amounts of avermectin B_{1a} present initially and with time, dissipation profiles of avermectin B_{1a}, and half-lives of avermectin B_{1a} after 1 week postapplication. Therefore, on the basis of these results, the fate of [^{3}H]avermectin B_{1a} was the same as that of the ring-labeled [^{14}C]avermectin B_{1a} on picked and field oranges. Furthermore, the fate of [^{3}H]avermectin B_{1a} was the same as that of [^{14}C]avermectin B_{1a} in animal metabolism studies (Maynard et al., 1985a,b), soil metabolism studies (Bull et al., 1984), and other plant metabolism studies (Bull et al., 1984). Therefore, the use of [^{3}H]avermectin B_{1a} in citrus metabolism studies provided results that were the same as those obtained with [^{14}C]-avermectin B_{1a}.

In conclusion, no differences were observed between the application rate on the fate of avermectin B_{1a} on citrus fruits. The ^{3}H-labeled avermectin B_{1a} produced the same results as [^{14}C]avermectin B_{1a} on citrus and is, therefore, a valid label to use. From these studies, the fate of avermectin B_{1a} was shown to be similar on both picked and field orange fruit. The picked fruit studies may lead to higher residue levels than would be expected in the field since the removal of volatile degradates was inefficient in this picked fruit procedure. However, the fate of avermectin B_{1a} was accurately investigated on picked citrus fruits. The similarity to the field fruit was attributed to the fact that avermectin B_{1a} appears to degrade on the fruit surface independent of whether oranges are picked or remain on the tree. The fate of avermectin B_{1a} may, therefore, be similar on many crops. This picked fruit procedure may be applicable for citrus fruit studies with other pesticides that are primarily degraded by non-plant-related mechanisms (i.e., photolysis, autooxidation, etc.).

Registry No. Avermectin B_{1a}, 65195-55-3.

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